CLAIMS

[1] A pharmaceutical composition for preventing or treating a Th1-mediated immune disease, which comprises as an active ingredient a substance capable of acting on the natriuretic peptide receptor guanylyl cyclase A to enhance the production of cyclic guanosine monophosphate.

[2] The pharmaceutical composition according to claim 1, wherein the Th1-mediated immune disease is selected from a disease due to graft rejection following transplantation, graft-versus-host disease caused by bone marrow (hematopoietic stem cell) transplantation, and an autoimmune disease.

[3] The pharmaceutical composition according to claim 2, wherein the autoimmune disease is selected from autoimmune hepatitis, chronic rheumatoid arthritis, insulin-dependent diabetes mellitus, ulcerative colitis, Crohn's disease, multiple sclerosis, autoimmune myocarditis, psoriasis, scleroderma, myasthenia gravis, multiple myositis/dermatomyositis, Hashimoto's disease, autoimmune hypocytosis (e.g., pure red cell aplasia, aplastic anemia), Sjogren's syndrome, vasculitis syndrome, and systemic lupus erythematosus.

[4] The pharmaceutical composition according to claim 3, wherein the autoimmune disease is Crohn's disease or multiple sclerosis.

[5] The pharmaceutical composition according to claim 1, wherein the substance capable of acting on the natriuretic peptide receptor guanylyl cyclase A to enhance the

production of cyclic guanosine monophosphate is a natriuretic peptide.

[6] The pharmaceutical composition according to claim 5, wherein the natriuretic peptide is atrial natriuretic peptide or brain natriuretic peptide.

[7] The pharmaceutical composition according to claim 6, wherein the atrial natriuretic peptide is of human origin.

[8] A method for treating a Th1-mediated immune disease, which comprises administering a substance capable of acting on the natriuretic peptide receptor guanylyl cyclase A to enhance the production of cyclic guanosine monophosphate.

[9] The method according to claim 8, wherein the Th1-mediated immune disease is selected from a disease due to graft rejection following transplantation, graft-versus-host disease caused by bone marrow (hematopoietic stem cell) transplantation, and an autoimmune disease.

[10] The method according to claim 9, wherein the autoimmune disease is selected from autoimmune hepatitis, chronic rheumatoid arthritis, insulin-dependent diabetes mellitus, ulcerative colitis, Crohn's disease, multiple sclerosis, autoimmune myocarditis, psoriasis, scleroderma, myasthenia gravis, multiple myositis/dermatomyositis, Hashimoto's disease, autoimmune hypocytosis (e.g., pure red cell aplasia, aplastic anemia), Sjogren's syndrome, vasculitis syndrome, and systemic lupus erythematosus.

[11] The method according to claim 10, wherein the autoimmune disease is Crohn's disease or multiple sclerosis.

[12] The method according to claim 8, wherein the

substance capable of acting on the natriuretic peptide receptor guanylyl cyclase A to enhance the production of cyclic guanosine monophosphate is a natriuretic peptide.

[13] The method according to claim 12, wherein the natriuretic peptide is atrial natriuretic peptide or brain natriuretic peptide.

[14] The method according to claim 13, wherein the atrial natriuretic peptide is of human origin.

[15] Use of a substance capable of acting on the natriuretic peptide receptor guanylyl cyclase A to enhance the production of cyclic guanosine monophosphate for the manufacture of a pharmaceutical composition for preventing or treating a Th1-mediated immune disease.

[16] The use according to claim 15, wherein the Th1-mediated immune disease is selected from a disease due to graft rejection following transplantation, graft-versus-host disease caused by bone marrow (hematopoietic stem cell) transplantation, and an autoimmune disease.

[17] The use according to claim 16, wherein the autoimmune disease is selected from autoimmune hepatitis, chronic rheumatoid arthritis, insulin-dependent diabetes mellitus, ulcerative colitis, Crohn's disease, multiple sclerosis, autoimmune myocarditis, psoriasis, scleroderma, myasthenia gravis, multiple myositis/dermatomyositis, Hashimoto's disease, autoimmune hypocytosis (e.g., pure red cell aplasia, aplastic anemia), Sjogren's syndrome, vasculitis syndrome, and systemic lupus erythematosus.

[18] The use according to claim 17, wherein the

autoimmune disease is Crohn's disease or multiple sclerosis.

[19] The use according to claim 15, wherein the substance capable of acting on the natriuretic peptide receptor guanylyl cyclase A to enhance the production of cyclic guanosine monophosphate is a natriuretic peptide.

[20] The use according to claim 19, wherein the natriuretic peptide is atrial natriuretic peptide or brain natriuretic peptide.

[21] The use according to claim 20, wherein the atrial natriuretic peptide is of human origin.

[22] A method for regulating the Th1/Th2 balance in the immune system, which comprises treating dendritic cells with a substance capable of acting on the natriuretic peptide receptor guanylyl cyclase A to enhance the production of cyclic guanosine monophosphate, and thereby polarizing T cells toward Th2-promoting phenotype.

[23] The method according to claim 22, wherein the substance capable of acting on the natriuretic peptide receptor guanylyl cyclase A to enhance the production of cyclic guanosine monophosphate is a natriuretic peptide.

[24] The method according to claim 23, wherein the natriuretic peptide is atrial natriuretic peptide or brain natriuretic peptide.

[25] The method according to claim 24, wherein the atrial natriuretic peptide is of human origin.